

ABSTRACTS OF CARDIOLOGY

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The Eisenmenger Syndrome or Pulmonary Hypertension with Reversed Central Shunt. P. WOOD. *Brit. med. J.*, 2, 701 and 755, 1958.

In this paper (which is the text of the Croonian Lectures for 1958) the author suggests that the condition of which a typical example was first described by Eisenmenger in 1897 and which has since become known as Eisenmenger's complex can be redefined in the light of modern knowledge as "pulmonary hypertension at systemic level, due to a high pulmonary vascular resistance (over 800 dynes per second per cm.⁵), with reversed or bidirectional shunt through a large ventricular septal defect (1.5 to 3 cm. across)." He defends the retention of the eponym for this condition, but points out that "a remarkably similar physiological situation occurs when any large communication between the two circulations is complicated by a pulmonary vascular resistance around systemic level" and lists 12 different types of anatomical abnormality that may present in this way. Since the exact anatomical diagnosis in such cases may be uncertain, he suggests that the term "Eisenmenger's syndrome" be used to embrace all of this group of conditions "when behaving physiologically like 'Eisenmenger's complex' proper." He describes the distinctive characteristics of the members of the group on the basis of an analysis of 127 cases of Eisenmenger's syndrome personally investigated, of which 15 came to necropsy, together with 38 cases collected from other sources, all of which were examined post mortem.

Of the various abnormalities with which Eisenmenger's syndrome may be associated the commonest are patent ductus arteriosus, ventricular septal defect, and atrial septal defect. Patients with atrial septal defect generally develop the syndrome, if at all, in adult life, the syndrome presenting at an average age of 35 years. Their high pulmonary resistance is apparently acquired and is by no means inevitable, for the pulmonary resistance usually remains low in spite of a lifelong heavy pulmonary flow. In contrast, patients with large interventricular and aorto-pulmonary communications usually develop the Eisenmenger syndrome early in life, for the right ventricle and pulmonary artery are inescapably exposed to systemic pressure. Their high pulmonary resistance is established at birth or in infancy.

Among the 53 cases in which necropsy was performed death was due to hæmoptysis in 29 per cent, attempted surgical cure in 26 per cent, congestive failure in 17 per cent, and presumed ventricular fibrillation in 14 per cent. The size of communication in cases of patent ductus arteriosus and ventricular septal defect was large compared with that usually found in similar cases without Eisenmenger's syndrome, whereas in cases of atrial septal

defect it was no bigger than in cases with a normal or low pulmonary vascular resistance. The histological changes in the pulmonary vessels are briefly described, and the protective effect of pulmonary stenosis is noted. Whether or not the aorta overrides the right ventricle is considered to be unimportant for the genesis of the syndrome.

The occurrence of a common physiological pattern in association with so many different types of anatomical defect is explained by the author as follows. In the normal child at birth the pulmonary resistance falls rapidly with the expansion of the lungs with air to a value roughly equal to that of the systemic circulation, its further fall to adult level being a gradual process associated with diminishing vasoconstrictor tone due to rising alveolar oxygen tension, involution of the muscular pulmonary arteries, and closure of the ductus arteriosus. This process is prevented in most cases of Eisenmenger's syndrome by the presence of a large communication which exposes the pulmonary arterial tree to systemic pressure, so that vasoconstrictor tone is maintained and atrophy of the muscular arteries is prevented. But in atrial septal defect no left-to-right shunt occurs for some time after birth so that pulmonary vascular resistance usually falls to normal. In a few cases it remains slightly raised and gradually increases further with the development of intimal fibrosis.

Surgical cure should be attempted only when it can be shown that the pulmonary resistance is not grossly raised and that closure of the defect will result in a fall in pulmonary pressure. Vasodilator drugs are useless, and medical treatment should aim at attempting to prevent secondary thrombotic lesions of the pulmonary vessels by means of anticoagulants.

(The essential feature of the Eisenmenger syndrome, the high pulmonary vascular resistance, is convincingly explained by the author, whose closely reasoned argument is supported by a wealth of well presented data. These lectures cannot readily be abstracted and should be read in full.)
J. A. Cosh

Late Results of Operations for Fallot's Tetralogy. M. CAMPBELL. *Brit. med. J.*, 2, 1175, 1958.

In this paper, the author reports the results obtained with subclavian—pulmonary anastomosis, pulmonary valvotomy or infundibular resection or both in 241 patients in whom Fallot's tetralogy was diagnosed.

"Good" and "very good" results were obtained with anastomotic operations in 130 cases. Recent reports on 126 of these patients showed that 8 had died, 17 had lost much or all of the improvement, and 101 were

"as good or nearly as good as they were." Of these 101, who were followed up for an average of $7\frac{1}{2}$ years 11 were well after 10 years, 10 after 9 years, 28 after 8 years, 36 after 7 years, and 16 after 6 years. The results of direct operations were good in 79 out of 111 patients, 70 remaining well for an average period of 5 years. After direct operations the annual death rate and the proportion of patients who lost all improvement were only about half as high as after the corresponding anastomotic operations.

Complications, especially cerebral abscess, of the incompletely corrected congenital defect caused over one-third of the deaths in the series. A further one-third were caused by heart failure under the load of an increased pulmonary flow. Closure of the anastomosis (with disappearance of the continuous murmur) or an apparent increase in the severity of pulmonary stenosis caused deterioration in many cases.

He emphasizes that none of these operations is curative, for the septal defect remains. Nevertheless, he considers that half the patients who obtain good results "will be reasonably well after 21 years, but the average age will be only about 31 years . . . Perhaps one-quarter will be able to carry on in this way until they are 50."

D. Emslie-Smith

The Surgical Treatment of Tetralogy of Fallot. SECTION ON CARDIOVASCULAR SURGERY, AMERICAN COLLEGE OF CHEST PHYSICIANS. *Dis. Chest*, **34**, 103, 1958.

This valuable report analyses information, supplied by 39 surgeons in various parts of the world in response to a questionnaire, concerning the results of surgical treatment in 4062 cases of Fallot's tetralogy. Unfortunately, inadequate data were provided in many cases, so that the findings in certain respects refer to only a part of the whole series, but the numbers available are large enough to give a good idea of the value of the various procedures undertaken.

In most cases closed techniques were employed, the standard operations being subclavian-pulmonary anastomosis (Blalock), aortico-pulmonary anastomosis (Potts), and direct pulmonary valvotomy or infundibular resection. In general the mortality and results of these procedures were similar. In cases in which resection of an infundibular obstruction was performed by an open technique (usually with a by-pass technique) the mortality was higher, but preliminary results in the survivors appeared to be excellent. The operative mortality (up to 3 months after operation) was 10.6 per cent in 515 cases treated by Pott's operation, 13.8 per cent in 1497 treated by Blalock's operation, 16.3 per cent in 238 treated by valvotomy, and 23.8 per cent in 67 treated by closed infundibular resection, while the corresponding figure for 122 cases treated by open repair was 38.5 per cent. Clinical results, classified simply as "good" or "poor" on an over-all assessment, were reported for 1681 survivors. The indirect anastomotic procedures (1409 cases) gave 85 to 87 per cent, closed pulmonary valvotomy or infundibulectomy (232 cases) 75 to 80 per cent, and open repair (40 cases) 100 per cent "good"

results. Among the other points mentioned in the report are the following: operative risk appears to be higher with increasing age (16 per cent mortality among 261 patients under 14 years, 23 per cent among 60 over 14 treated by various techniques); bacterial endocarditis or brain abscess occurred in 1.5 per cent of 901 survivors; the blood count returned to normal in only 23 per cent of 386 cases; absence of a pulmonary artery that could be used in establishing an adequate shunt was reported in 4 per cent of 1033 cases.

From a critical review of the figures available the conclusion is reached that a patient with Fallot's tetralogy facing operation has a 61 per cent chance of obtaining a "good" result and a 39 per cent chance of death or a "poor" result.

T. Holmes Sellors

A Follow-up Study of 250 Patients Subjected to Surgical Treatment of Patent Ductus Arteriosus. N. HELSINGEN, O. HUSOM, and L. EFSKIND. *Thorax*, **13**, 210, 1958.

The follow-up results in 250 patients operated on at Rikshospitalet, Oslo, for patent ductus arteriosus (P.D.A.) between 1944 and 1956 are discussed. Of these patients, 23 per cent of whom were male, 110 (44%) had no symptoms when first seen, 58 had had one or more attacks of severe respiratory infection, and most of the adults had symptoms of cardiac embarrassment. In at least 21 cases there were other congenital heart lesions, such as atrial septal defect. The youngest patient in this series was aged 14 months and the oldest 40 years. Approach was by dorso-lateral thoracotomy, and in most cases simple ligation with silk was performed. The mortality was 1.2 per cent.

At follow-up after periods varying from 6 months to 13 years, all the patients being traced, many of the children showed an improvement, even though considered symptomless before operation. In 4 cases (2 adults) a murmur indicating recanalization was heard—a recurrence rate of 1.6 per cent.

The authors consider that in most cases of P.D.A. simple ligation is satisfactory and the risk of recurrence small except in those with a giant duct and in older patients.

M. Meredith Brown

Percutaneous Left Ventricular Puncture with Catheterization of the Aorta. H. A. FLEMING, E. W. HANCOCK, B. B. MILSTEIN, and D. N. ROSS. *Thorax*, **13**, 97, 1958.

The authors describe the method of catheterization of the aorta used at the Brompton and Guy's Hospitals, London, in the investigation of cases of aortic valvular stenosis. Following percutaneous puncture of the left ventricle a fine plastic catheter is passed into the aorta through the puncture needle, which is of slightly larger bore than usual. In addition to allowing measurement of the systolic pressure gradient across the aortic valve this procedure enables the detection of subvalvular stenosis to be made by noting the pressure changes occurring during withdrawal of the catheter from the aorta back into the ventricle.

The authors' experience in the first 36 cases (out of

115) are described, with details of the technique. In 28 the aorta was entered successfully and pressure tracings of generally good quality were obtained, 5 cases of subaortic stenosis being identified. Although the method may thus be helpful, it appears to be more dangerous than left ventricular puncture alone with a smaller gauge needle. There were 2 deaths in the series, both associated with hæmopericardium and blood loss. Cardiac tamponade, which subsided uneventfully, was diagnosed in a third case, while 2 more patients developed sudden severe chest pain, transient collapse, and a pericardial friction rub some days after the puncture. A small number of patients also experienced pericardial pain during the procedure. Because of this relatively high incidence of complications it is suggested that the investigation be restricted to those patients in whom subaortic stenosis is suspected, and that ischæmic heart disease and myocardiopathy be regarded as contraindications.

S. G. Owen

Pulsus Alternans in Aortic Stenosis. Hemodynamic Observations in 50 Patients Studied by Left Heart Catheterization. T. COOPER, E. BRAUNWALD, and A. G. MORROW. *Circulation*, 18, 64, 1958.

In 28 patients with acquired (rheumatic) aortic stenosis (ranging in age from 30 to 55 years) and 22 patients with congenital aortic stenosis (age range 6 to 36 years), the left ventricular pressure and arterial pressure—the latter either in the central aorta by means of retrograde arterial catheterization or in the femoral or brachial artery by percutaneous puncture—were recorded simultaneously with the electrocardiogram. In 46 of the patients left heart catheterization was carried out by the transbronchial route and in the other 4 by percutaneous puncture through the anterior chest wall.

In 15 of the 28 patients with acquired aortic stenosis persistent pulsus alternans was present in the left ventricular pressure tracing, the average difference in pressure between the large and small pulses being 15 (range 2 to 43) mm. Hg. Patients with persistent pulsus alternans had a significantly higher left ventricular systolic pressure and a higher aortic valve gradient than those without, but the hæmodynamic parameter affording the best separation between these two groups was the product of left ventricular systolic pressure multiplied by heart rate, which is a measure of the relative oxygen requirements. From this the inference is drawn that a disproportion between the oxygen requirement of the heart and the supply of oxygen available to the heart can so alter cardiac contractility that alternation results. However, none of the 22 patients with congenital aortic stenosis showed persistent pulsus alternans and in consequence it is concluded that some other factor may play a part in the genesis of pulsus alternans. Among the 15 patients with the acquired lesion who were operated on, only 6 of the 11 with persistent pulsus alternans survived, whereas of the 4 without pulsus alternans, 3 survived. In contrast the 12 patients with congenital aortic stenosis subjected to operation all survived.

A. Schott

Mitral Stenosis with Jet Regurgitation. A. A. F. PEEL. *Scot. med. J.*, 3, 422, 1958.

In this paper, the author defines and describes the clinical, radiographic, phonocardiographic, and hæmodynamic characteristics whereby cases of jet regurgitation through a stenosed mitral valve may be differentiated from cases of free mitral regurgitation with little or no stenosis. Although in most cases of the former type the physical signs are predominantly those of mitral stenosis and the condition is recognized only at operation, some of them present with signs of mitral regurgitation predominating and, unless differentiated from cases of free regurgitation, will be rejected for operation. Since free valvotomy sometimes leads to dramatic improvement in these cases, their clinical recognition is important. Seven such cases are described by the author in detail and the findings analysed in a series of tables. He also describes the operation findings and results in 5 of these cases and 11 others in which the clinical signs were predominantly those of mitral stenosis.

A. I. Suchett-Kaye

Bronchial Constriction in Mitral Stenosis Studied by Pulmonary Function Tests (In English). K. BÜLOW. *Cardiologia*, 33, 211, 1958.

The author reports that pulmonary function studies undertaken at the University of Lund, Sweden, on 30 patients with mitral stenosis and 23 controls have confirmed the presence of bronchial constriction as a factor aggravating respiratory incapacity in patients with mitral stenosis. Determination of the maximum expiratory flow rate and the one-second vital capacity were the most useful tests in demonstrating improvement of function after the subcutaneous injection of adrenaline, but the simple measurement of vital capacity also showed a significant increase in this parameter. No improvement after administration of adrenaline was observed in patients with Grade-IV mitral valvular disease.

T. Semple

The Surgical Treatment of Mitral Insufficiency by Mitral Annuloplasty. L. D. GUIDRY, J. A. CALLAHAN, H. W. MARSHALL, and F. H. ELLIS. *Proc. Mayo Clin.*, 33, 523, 1958.

During 1958 at the Mayo Clinic 6 patients aged 23 to 49 years suffering from pure mitral regurgitation were successfully treated by narrowing of the mitral annulus. The operation was performed with the help of extracorporeal circulation without cardiac arrest.

Clinical details and the results of catheter studies for each of the patients are reported and tabulated. A competent valve with no significant stenosis was achieved in each case by this procedure. Transient neurological disturbance seen in 2 patients was believed to be due to air embolism and was the only operative complication encountered. Follow-up catheter studies suggested the recurrence of slight incompetence in one patient, but there was remarkable symptomatic improvement in all 6 patients and the authors consider that the operation of

mitral annuloplasty "offers considerable promise for the correction of pure mitral insufficiency."

C. A. Jackson

drug for the relief of angina, the effects being due, in part at least, to cerebral stimulation, but that the side-effects limit its therapeutic use.

Gerald Sandler

Anastomosis in the Coronary Circulation. W. LAURIE and J. D. WOODS. *Lancet*, 2, 812, 1958.

In an attempt to determine whether or not there are functional anastomoses between the coronary arteries and whether, as has been suggested, these increase with age, the authors, at the Edendale Hospital, Natal, examined normal and diseased hearts from Bantu patients. In all 200 hearts were examined, 150 of which proved suitable for study. The procedure consisted in the injection of one coronary artery with a gelatin suspension containing lead phosphate. This radio-opaque mass was about seven times more viscid than heparinized blood, and the particle size of the lead phosphate was not less than 35μ , so that none of the material should have passed the capillaries. With the heart immersed in saline at 45°C . one coronary artery was injected at a pressure of 150 mm. Hg for 20 minutes. The heart was then cooled until the injection mass hardened. The heart was "unrolled" by Schlesinger's technique and radiographs obtained of the coronary circulation. No difference in the anastomoses was observed whether the right or left coronary artery was injected. Some degree of filling of the uninjected artery was seen in 48 out of 87 normal hearts, and in 12 of these filling was complete. Some degree of anastomosis was found in 13 out of 46 healthy subjects under the age of 4 years and in 35 out of 41 over that age. However, of 63 diseased hearts, 40 showed no demonstrable anastomoses.

It is suggested that the majority of healthy people "have a functionally important inherited coronary anastomotic blood-supply (and this) probably protects some against ischaemic heart-disease and may modify ischaemic heart-disease in others."

C. Bruce Perry

Iproniazid (Marsilid) in Angina Pectoris. A. M. MASTERS, *Amer. Heart J.*, 56, 570, 1958.

The value of iproniazid in the relief of angina was studied in 74 patients. Each received 50 mg. of iproniazid 3 times a day with 25 mg. of pyridoxine with each dose of iproniazid to minimize side-effects, the duration of treatment being one week to five months.

There was complete relief of angina in 13 patients, considerable improvement in 28 patients, "doubtful" change in 4, and no change in 29. The improvement began within 3 to 10 days and lasted up to 2 weeks after the drug was discontinued. Improvement was mainly subjective only, since in a number of patients obtaining relief from angina there was no change in the abnormal electrocardiogram or in the response to exercise tolerance tests. Side-effects included gastro-intestinal disturbances (mainly constipation and dry mouth), disturbances of micturition and impotence, and dizziness. In 57 of the patients iproniazid had to be discontinued because of the severity of these side-effects.

The author concludes that iproniazid is a most effective

Use of Iproniazid in Ischaemic Angina Pectoris. M. K. TOWERS and P. WOOD. *Brit. med. J.*, 2, 1067, 1958.

Iproniazid was found by Cesarman to relieve anginal pain in a patient to whom it was given for its euphoriant action. A trial of this drug in 40 patients with severe disabling angina is reported from the Institute of Cardiology, London.

It is concluded that iproniazid is very effective in angina, and that pain may be relieved in spite of a deterioration in the ECG. The drug blocks pain, but does not influence the natural course of coronary arterial disease. Theoretically, it may increase the risk of infarction by allowing the patient to be more active and therefore anticoagulants should be given in addition, but because of this risk and the side-effects it should be given only to patients with severe disabling angina.

David Phear

The Influence of Anticoagulant Therapy on the Occurrence of Cardiac Rupture and Hemopericardium following Heart Infarction. I. A Study of 98 Cases of Hemopericardium (81 of Them Cardiac Ruptures). S. AARSETH and H. F. LANGE. *Amer. Heart J.*, 56, 250, 1958.

II. A Controlled Study of a Selected Treated Group Based on 1044 Autopsies. H. F. LANGE and S. AARSETH. *Amer. Heart J.*, 56, 257, 1958.

This series of 89 cases of hæmopericardium found at necropsy on 1229 patients, representing 94 per cent of the total of 1303 dying of myocardial infarction at Ullevål Hospital, Oslo, during the period 1945-56, is thought by the authors to be the largest series of such cases published up to the present. The incidence of hæmopericardium (7.3%) among these deaths from myocardial infarction (in 81 of which (6.6% of the total) it was accompanied by cardiac rupture) is in accord with previous findings. In all cases the rupture occurred in the left ventricle. The age distribution of the patients with hæmopericardium corresponded with that for all cases of infarction, the majority being between 60 and 80 years.

In the second paper the results in 69 cases of myocardial infarction considered to have had adequate anticoagulant treatment during life were compared with those in 72 control cases, comparable as to age and sex, which had received no anticoagulants. Intracardiac mural thrombi were found less frequently (4 cases, or 6%) in the treated group than in the untreated group (8 cases, or 11%), but in the treated group hæmopericardium (1 case) and myocardial rupture (14 cases) was nearly twice as frequent as in the control group (8 cases out of 72, of which 7 were in women). It is suggested that anticoagulant therapy in cases of myocardial infarction "may contribute to a weakening of the affected area of the myocardium, with consequent rupture."

P. Hugh-Jones

Relation of Hemorrhage and Thrombosis to Prothrombin during Treatment with Coumarin-type Anticoagulants.

H. S. SISE, S. M. LAVELLE, D. ADAMIS, and R. BECKER. *New Engl. J. Med.*, **259**, 266, 1958.

With long-term anticoagulant treatment it is not unusual for hæmorrhage to occur in one patient and thrombosis in another, although the "Quick time" (defined as the clotting time of the plasma to which tissue thromboplastin and calcium have been added) is the same in both patients. The factors affected by the coumarin-type of anticoagulant are known to be prothrombin, proconvertin, Stuart factor, and plasma thromboplastin component. Decreases in the first three factors lead to a prolongation of the Quick time, but a decrease in the plasma thromboplastin component does not. The authors suggest that the prothrombin time gives a more accurate distinction between the hæmorrhagic and thrombotic states and is a more reliable indicator of over- or undertreatment with anticoagulants than the Quick time.

It was found that the degree of depression of prothrombin concentration and of the Quick time differed sufficiently in half the cases to indicate that the latter could not be relied upon to estimate prothrombin. Correlation of the occurrence of hæmorrhage and thrombosis with the prothrombin concentration was good, but that with the Quick time was poor. The authors recommend that both the Quick time and the prothrombin level should be determined in patients under treatment for a long time. The therapeutic range is indicated by a prothrombin concentration of 12 to 25 per cent when the Quick time is under 40 seconds.

D. Preiskel

Thrombolysis with Fibrinolysin (Plasmin)—New Therapeutic Approach to Thromboembolism. K. M. MOSER. *J. Amer. med. Ass.*, **167**, 1695, 1958.

Writing from Georgetown University Medical Center, Washington, D. C., the author describes the results of a pilot study of the effects of intravenous infusions of purified lyophilized fibrinolysin prepared from human plasma ("actase") given over 2 to 4 hours to 52 patients suffering from various thrombo-embolic states, the amount of the infusion being between 30,000 and 60,000 fibrinolytic units.

In 15 patients with deep venous thrombophlebitis, 13 of whom also received anticoagulant therapy, in 4 out of 8 patients with pulmonary embolism from an unknown source, the treatment led to rapid resolution of electrocardiographic anomalies, and relief of symptoms. Of 14 patients suffering from cerebral thrombosis (only 4 of whom received the infusion given within 24 hours of onset), 7 showed some degree of objective improvement within 24 hours after infusion. Lastly, of 7 patients with superficial thrombophlebitis, no benefit was apparent in 3 who had had symptoms for more than 7 days, but in the other 4 direct evidence of thrombolysis by the treatment was observed. Owing to the danger of further thromboses the author suggests that anticoagulant drugs should be given concurrently with actase.

R. Wyburn Mason

The Changes in Concentration of Cholesterol in the Serum of Hypertensive Patients during Antihypertensive Therapy. Q. B. DEMING, M. E. HODES, A. BALTAZAR, J. G. EDREIRA, and S. TOROSDAG. *Amer. J. Med.*, **24**, 882, 1958.

The serum cholesterol level in 21 patients with advanced hypertension was determined at different times before and during treatment with reserpine, hydrallazine, and the ganglion-blocking drugs chlorisondamine and mecamlamine. Therapy was given for at least 4 weeks, multiple cholesterol determinations being carried out during that time. Since wide variations in the serum cholesterol level were found during periods when medication, diet, and activity remained constant, the authors do not accept a change of less than 10 per cent in the results after statistical analysis as significant.

The results are derived from observations on 12 patients treated with oral reserpine alone, 7 with oral hydrallazine alone, 7 with hydrallazine plus reserpine, 5 with chlorisondamine (3 of the patients being given it both orally and parenterally in separate courses), and 4 with mecamlamine. In those given reserpine, although the serum cholesterol level tended to fall, only 4 patients showed a change of more than 10 per cent with a mean blood pressure fall of 14 mm. Hg; however, in those treated with hydrallazine the cholesterol level fell by over 10 per cent in 5 of the 7 patients (mean fall 28 mg. per 100 ml. for an average fall in blood pressure of 24 mm. Hg). Treatment with chlorisondamine was regularly accompanied by a significant and sustained fall in the serum cholesterol level (average decrease 55 mg. per 100 ml. with an average fall in blood pressure of 23 mm. Hg). All of the 4 patients given mecamlamine showed a significant fall in serum cholesterol level (average 81 mg. per 100 ml. for an average fall in blood pressure of 24 mm. Hg). The combined data from all treatment groups, representing 38 courses of treatment, showed a positive correlation coefficient of 0.51 ($P=0.01$) between the change in serum cholesterol level and the change in blood pressure. The authors discuss the possible meaning of this relationship and conclude that their findings confirm other reports that serum cholesterol concentration decreases during therapy with hydrallazine or ganglion-blocking drugs, but not with reserpine.

P. Hugh-Jones

The Pathogenesis and Treatment of Hyponatremia in Congestive Heart Failure. R. E. WESTON, J. GROSSMAN, E. R. BORUN, and I. B. HANENSON. *Amer. J. Med.*, **25**, 558, 1958.

Although there are probably numerous factors concerned in the development of œdema in congestive heart failure, sodium retention is now accepted as the primary cause of the œdema. However, in some cases of congestive failure water may be retained in excess of sodium and the urine has been reported to contain an increased amount of antidiuretic material.

Further detailed metabolic studies of water retention in congestive failure were therefore undertaken on 3 patients suffering from chronic cardiac failure due to rheumatic heart disease. These patients, who were

maintained on a low-sodium diet (12 to 15 mEq. daily) together with digitalis and bed rest, were studied during a period of increased cardiac failure and increased oedema; the cardiac deterioration was due to "escape from digitalization," a severe respiratory infection, and potassium depletion with resulting digitalis intoxication respectively. In all 3 cases the phase of cardiac deterioration was found to invoke an antidiuretic mechanism, resulting in retention of water in excess of sodium, an increase in the oedema, and a fall in the serum sodium level, though without external sodium loss. Infusion of a concentrated salt solution did not produce improvement, but with improving cardiac function a water diuresis with decrease in the oedema occurred. In one patient the water retention was reproduced by administration of vasopressin.

The successful treatment of water retention depends on the improvement of cardiac function. This may be possible by increasing the dose of digitalis or correcting potassium depletion. Concomitant pulmonary infarction, infection, or a recurrence of rheumatic fever are other possible treatable factors. Progressive terminal myocardial deterioration is the commonest (and irreversible) cause of the oedema, though something may be achieved here by careful restriction of water intake.

David Phear

Electrocardiogram in Chronic Severe Anemia. L. M. SANGHVI, S. N. MISRA, K. BANNERJI, and K. D. GUPTA. *Amer. Heart J.*, 56, 79, 1958.

Electrocardiographic abnormalities were found in 85 out of a series of 100 patients with anaemia of at least 3 months' duration, but without any other evidence of cardiovascular disease, who were examined at Sawai Mau Singh Hospital, Jaipur, India. The ages of the patients ranged from 8 to 50 years, and their haemoglobin level ranged from 2 to 8 g. per 100 ml., with an average of 2.6 g. per 100 ml. Heart failure was present in 30 cases. Most of the abnormalities found have been reported by other workers.

There was a fairly close, though by no means constant, relationship between the incidence of the abnormalities, the severity of the anaemia, and the size of the heart. In 36 cases the abnormalities persisted after cure of the anaemia. This did not appear to be related to age, 26 of the patients being under 30, but it was associated with persistent cardiac enlargement in most cases.

William A. R. Thomson

Radiological Observations on 33 Cases of Primary Interstitial Myocarditis during an Outbreak in the Haifa Area. J. MUNK and K. T. LEDERER. *J. Fac. Radio.*, 9, 195, 1958.

In 1956-57, 61 cases of primary interstitial myocarditis in children aged 6 months to 3½ years of age were seen in the Rambam Government and Rothschild Hospitals, Haifa, Israel. X-ray examination was carried out in 33 cases only, the remaining patients being *in extremis* or dead on admission. Of these 33, 22 died, necropsy being performed on 21.

The predominant radiobiological abnormality was generalized cardiac enlargement with passive pulmonary congestion, including, in some cases, lymphatic engorgement. There was a trace of pleural fluid in all except 5 cases, and cardiac pulsations were diminished on fluoroscopy in 16. Marked emphysema, which was probably due to concomitant diffuse interstitial pneumonia, a fairly common condition in Israel, was present in some cases. Discussing the differential diagnosis from interstitial pneumonia, the authors stress the importance of cardiac enlargement and Kerley's B lines in the diagnosis of myocarditis, and of emphysema of the periphery of the lungs in pneumonia.

D. E. Fletcher

A Study of Heart Disease in 108 Patients Dying with Portal Cirrhosis. J. H. LUNSETH, E. G. OLMSTEAD, and F. ABBOD. *A.M.A. Arch. intern. Med.*, 102, 405, 1958.

Details are given of the condition of the cardiovascular system as seen at necropsy on 108 patients who died of portal cirrhosis. The ages of the patients, 26 women and 82 men, ranged from 26 to 83 years, with a mean of 57 years. In 99 cases determination of the heart weight showed that this was greater than in a comparable series without cirrhosis. In 52 there was associated heart disease of the following types: arteriosclerotic in 26, hypertensive in 9, and valvular in 5; in the remaining 12 the cardiac hypertrophy could not be attributed to any of the usual causes and these are considered in considerable detail.

All 12 patients were alcoholic and presented a clinical picture of both heart and liver disease. Clinically, there were congestive cardiac failure, unexplained cardiomegaly, apical systolic murmurs, and tachycardia accompanied by premature auricular and ventricular beats or intraventricular conduction defects. Pathological studies indicated that cardiac hypertrophy was significantly more frequent in early and moderately advanced cirrhosis than in far-advanced disease. The authors suggest that a heart that may have been hypertrophic in the early stages of cirrhosis may revert to a normal weight in the advanced stages. It is concluded that patients with portal cirrhosis are prone to develop an idiopathic type of heart disease associated with the cirrhosis, in addition to the common types of heart disease.

J. Warwick Buckler

Valvular Pulmonary Stenosis with Intact Ventricular Septum. Clinical and Physiologic Response to Open Valvuloplasty. S. G. BLOUNT, J. VAN ELK, O. J. BALCHUM and H. SWAN. *Circulation*, 15, 814-826, 1957.

The authors consider that for the correction of valvular pulmonary stenosis with intact ventricular septum open valvuloplasty performed by the transarterial approach under hypothermia with circulatory occlusion gives good results. In a series of 38 patients so treated (and reported from the University of Colorado School of Medicine, Denver), of whom 10 were cyanosed, there were 2 deaths, both in cyanosed patients. The results of pulmonary arterial and right ventricular pressure measurements and oxygen saturation levels are given in detail for the first

25 patients, and only these patients are included in the analysis.

Of the 10 who had a defect of the atrial septum, 8 were cyanosed and had limited exercise tolerance; the latter was relieved in all, and in 4 cases the cyanosis was improved by the operation. Of the 15 patients with an intact atrial septum, 7 had no symptoms; in the other 8, fatigue and exercise tolerance were improved after operation.

In the younger patients the valve was usually a symmetrical elongated cone, but in the 3 adults in the series an asymmetrical fusion of thickened cusps was more common. A preoperative systolic pressure gradient ranging from 38 to 175 mm. Hg was reduced to less than 20 mm. Hg in the first 3 patients, in whom the valve was partially excised, and in 14 of the others. A murmur of pulmonary regurgitation was heard in these first 3 patients and in 8 others, but was not of haemodynamic significance. Among the possible causes of a residual pressure gradient is hypertrophy of the crista supraventricularis of the right ventricular outflow tract; removal of a distal obstruction unmasks a proximal one. In 6 patients moderate pulmonary hypertension developed following operation, but the cause of this could not be ascertained. The authors consider that this transarterial operation carries a lower mortality than that by the transventricular approach. Where there is an intact atrial septum the approach should be through the pulmonary artery. In the presence of a septal defect and right-to-left shunt the valve should be treated first, but if a left-to-right shunt is present the defect should be closed first and the valvuloplasty then performed at the same time if the patient's condition permits.

M. Meredith Brown

Atrial Septal Defect and its Surgical Treatment. D. E. BEDFORD, T. HOLMES SELLORS, W. SOMERVILLE, J. R. BELCHER, and E. M. M. BESTERMAN. *Lancet*, 1, 1255-1261, 1957.

The authors review their experience of 100 cases of atrial septal defect seen at the Middlesex Hospital, London, since surgical treatment of this abnormality has been undertaken there. It is important to exclude an ostium primum defect in patients submitted for surgery, 13 of this type being found in the series under review. This diagnosis is suggested by early cardiac enlargement, physical underdevelopment, and pulmonary hypertension with evidence of left ventricular hypertrophy.

Atrial septal defect causes serious cardiovascular change after the age of 20, and over 40 there is gross cardiac enlargement in half the cases. The authors are of the opinion that an uncomplicated fossa-ovalis defect with a shunt of 2:1 or more should be closed as soon as the diagnosis is established, and preferably before the age of 20 even in the absence of cardiac symptoms. Contraindications to surgery are ostium-primum defect, a reversed shunt, obstructive pulmonary hypertension with high vascular resistance, systemic hypertension, and advanced age and poor general condition.

During the past year 40 patients between the ages of 6 and 55 years have been operated upon. All these had fossa-ovalis defects separated from the tricuspid valve,

though some of the defects were very large, extending to the inferior caval orifice in 9 cases and to the superior caval orifice in 2. The defect was closed under direct vision with the patient anaesthetized by a modification of Swan's surface-cooling technique. Rewarming was started immediately on removal from the cold bath, an oesophageal temperature of 30° C. at the time of circulatory arrest being regarded as the ideal. Neostigmine injected into the coronary circulation is regarded as an important protection against ventricular fibrillation. In this way at least 7 and probably 10 minutes of circulatory arrest can be permitted. The anatomy of the defect is assessed and the mitral, tricuspid, and pulmonary valves are explored digitally through the right atrium. After establishing circulatory arrest the atrium is opened and the defect is closed with a continuous suture reinforced with one or two interrupted stitches. An important difficulty occurs in the case of low defects astride the vena caval orifice.

In the 40 cases operated upon there was only one death. This occurred during a second operation to correct central cyanosis due to inclusion of the inferior vena caval orifice in the left atrium. One patient is waiting for correction of this anomaly, and one, as already described, had the anomaly corrected immediately after its creation. The other cases are satisfactory and 12 of them have been recatheterized. In 7 of these the findings are completely normal; in 3 there is probably an insignificant shunt, and in 2 the shunts are appreciable.

The authors conclude that uncomplicated fossa-ovalis defects can be closed under hypothermia with little operative risk, particularly if the operation is performed before the age of 20.

[This is an important paper to which full justice cannot be done in an abstract. The original should therefore be consulted by those interested in the subject.]

A. M. Macarthur

Treatment of Heart Disease. A Clinical Physiologic Approach. By HARRY GROSS, M.D., F.A.C.P., and ABRAHAM JEZER, M.D. Pp. 549 and x. 91s. Philadelphia and London: W. B. Saunders Co.

Although, as the authors state in the preface, this book is a "presentation of therapy in heart disease based upon sound physiological principles", it includes many sections on the pathogenesis and clinical features of the various types of heart disease. The book has been clearly devised to provide practical help for the physician and after a full discussion of the various methods of treatment that are available, the authors present, often in summary form, their own views on management. Although the book is very up-to-date, the authors are conservative in their selection of methods of treatment, sometimes unexpectedly so as for example in the section on anticoagulants in myocardial infarction when they state that only a small group of patients benefit from the use of these drugs and "for the present, routine anticoagulant treatment should be limited to exceptional situations". There is a useful appendix of diets, menus, and recipes particularly for low salt and reducing diets. This book can be thoroughly recommended.

Graham Hayward